

Prevention

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bulletin

Save Hearts in Arizona Registry & Education

Sudden Cardiac Arrest: "How to Survive in '05"

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INCIDENCE

Heart disease is the number one killer in the United States. Every day, more than 2,600 Americans die from cardiovascular disease, which amounts to 1 death every 33 seconds.

Sudden out-of-hospital cardiac arrest is the leading cause of death and disability and a leading source of health care cost in the United States. However, unlike many other epidemics in health care, out-of-hospital cardiac arrest caused by ventricular fibrillation has an effective, proven treatment called defibrillation.

PATHOPHYSIOLOGY

Most of these deaths from sudden cardiac arrest occur with little or no warning. The most common cause of sudden cardiac arrest is a disturbance in the heart rhythm called ventricular fibrillation. Ventricular fibrillation (VF) is a disorganized, chaotic heart rhythm where the heart loses its ability to effectively pump oxygen-carrying blood to the brain and other vital organs.

If the brain and heart do not receive adequate blood flow, within a few minutes they start to shut down and cell death ensues. Specifically, if blood flow is not immediately restored to the brain, irreversible brain death occurs.

For every minute that goes by when a person remains in ventricular fibrillation, the chances of resuscitation decrease by approximately 10 percent. After 10 minutes, the chances of successful resuscitation are near zero.

In coronary care units, most people who experience ventricular fibrillation survive, because defibrillation is performed almost immediately.

The situation is just the opposite when cardiac arrest occurs outside a hospital, unfortunately the more common setting. Unless defibrillation is performed within the first few minutes after the onset of ventricular fibrillation, the chances for reviving the person (resuscitation) are very poor. The survival from out-of-hospital cardiac arrest in many large urban cities is less than 1%. However, some cities that have put much time and energy into public education on identifying cardiac arrest victims, bystander CPR, and the use of Automated External Defibrillators (AEDs) have documented greater success. In certain cities, survival to hospital discharge for cardiac arrest victims presenting with ventricular fibrillation has been as high as 40%.

Cardiopulmonary resuscitation, usually called CPR, provides temporary artificial breathing and circula-

tion. It can deliver a limited amount of blood and oxygen to the brain until a defibrillator becomes available. However, defibrillation is the only effective way to resuscitate a victim of ventricular fibrillation.

CHAIN OF SURVIVAL

CPR is one link in what the American Heart Association calls the Chain of Survival. The Chain of Survival is a series of actions that, when performed together, give the cardiac arrest victim the greatest chance of survival.

- ★ Early access: When an emergency is recognized, the first link in the Chain of Survival is early access. This means activating the emergency medical services, or EMS, system by calling 911. ("911" does not work in every community. Be sure to check your local directory, and know the correct emergency telephone number in your community.)
- ★ CPR: The second link in the Chain of Survival is to perform CPR until a defibrillator becomes available.
- ★ Early defibrillation: The third and most critical link in the Chain of Survival for a victim of ventricular fibrillation is early defibrillation.

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"Early" means up to 5 minutes after collapse.

- ★ **Early Advanced Life Support:** The last link in the Chain of Survival is early advanced life support. This is provided by experienced medical personnel such as paramedics, nurses, and doctors. Advanced life support includes administering medications and using advanced oxygen delivery techniques to resuscitate a person.

LACK OF BYSTANDER-INITIATED CPR

In the absence or delay of early defibrillation, bystander initiated chest compressions is essential for improved survival from cardiac arrest. A meta-analysis published in 1991 of 17 studies showed that individuals receiving bystander CPR were 4.5 times more likely to survive.

So why do we have such dismal survival rates?

The first problem contributing to the dismal survival rates from out-of-hospital cardiac arrest is the lack of bystander initiated CPR. Although the majority of out-of-hospital cardiac arrests are witnessed, only one in five receive bystander CPR. However, when surveyed on whether they would perform chest compression only CPR without mouth-to-mouth ventilation, 68% of those surveyed would do so. When asked why they were reluctant to perform CPR, over 80% confided they were afraid of contracting an infectious disease.

Research in the swine model has clearly shown that the most critical component of cardiopulmonary resuscitation is properly done, forceful, fast chest compressions. Chest compressions performed in this method result in the highest cerebral perfusion pressure and the greatest survival rates.

The supreme importance of chest compressions and minimizing their interruptions in adult cardiac arrest victims has lead many leading resuscitation experts to rename the old maxim Airway, Breathing, Circulation

(ABC) to Circulation, Breathing, Airway (CBA).

Additionally, the complexity of current basic life support measures with specific ratios of chest compressions and mouth breathing frequently confuses potential rescuers. This confusion often results in bystanders doing nothing due to fear of performing the steps in the wrong sequence or inadequately.

Current scientific evidence suggests that properly done continuous chest compression only CPR is equally if not more beneficial to cardiac arrest victims. Additionally, chest compression only CPR is far easier for laypersons to learn, remember and perform.

This, along with the real life fact that most people are unwilling or physically unable to do mouth to mouth breathing, is the reason for the SHARE Program decision to endorse cardiac resuscitation algorithms incorporating continuous chest compression only CPR.

AUTOMATED EXTERNAL DEFIBRILLATORS

In the mid-1980s, a new generation of computerized defibrillators was introduced called Automated External Defibrillators, or "AEDs" for short. These devices were capable of interpreting a person's heart rhythm and automatically delivering a defibrillation shock with only minimal input from the operator. For the first time, EMS personnel such as basic emergency medical technicians (EMTs) were able to provide the life-saving technique of defibrillation without having to interpret ECG rhythms.

As AEDs began to be placed in more and more "basic life support" ambulances (those not staffed by more advanced paramedics), the survival rates for out-of-hospital cardiac arrest began to rise. However, the problem of getting the defibrillator to the victim in less than 10 minutes remained a challenge.

The next step in reducing the amount of time it took to get a defibrillator to a cardiac arrest victim came



with the recognition that the police are often the first to arrive at the scene of a medical emergency, ahead of an EMS unit.

- ◆ With this knowledge, some EMS systems began to train and equip police officers to provide defibrillation with AEDs.
- ◆ This allowed defibrillation to be performed sooner, often before an ambulance arrived.
- ◆ The use of AEDs by law enforcement personnel has begun to have a significant impact in resuscitating victims of sudden cardiac arrest.

SHARE - SAVE HEARTS IN ARIZONA REGISTRY & EDUCATION

The origins of the SHARE Program are with Lani Clark at the University of Arizona Sarver Heart Center as a Regional Program. Clark's collaboration with Dr. Ben Bobrow in January 2005 resulted in the evolution of the SHARE Program into a statewide initiative now under the direction of the Bureau of Emergency Medical Services at the Arizona Department of Health Services.

The long-term goal of the SHARE Program is for Arizonans to have the highest survival rate in the world for out-of-hospital cardiac arrest. The Program has set out to attain that goal with a diverse approach including:

1. Public education that seeks to greatly advance the awareness of the general public in recognizing the signs and symptoms and immediate actions to take in the event of a cardiac arrest. Public

education is being done through community teaching, mass media, and via the SHARE website at www.AZSHARE.GOV.

2. Working closely with EMS agencies, SHARE is scientifically determining the most efficacious algorithms for prehospital cardiac resuscitation. Nationally, some cities track their cardiac arrest survival as a measure of their EMS Systems efficiency; however Arizona is unique in its ability to do this as a State. To accomplish this, SHARE maintains a Statewide, secure registry of EMS data from cardiac arrest victims and tracks cardiac arrest survivors for the rest of their lives with a yearly quality of life survey.
3. SHARE has developed a registry of AEDs throughout Arizona. SHARE provides training, QI measures, and the required Medical Direction for private and public entities to have AEDs.
4. This AED registry is yielding tremendous insight into who, where, and how AEDs are being used in Arizona. The AED registry also provides a method for contacting AED owners in the case of an FDA recall or other issues of importance.
5. The SHARE Program seeks new and unique methods to train first responders such as Police, Security Officers and community response teams who may perform the lifesaving defibrillation if on scene prior to EMS.
6. The Arizona Stroke Prehospital Identification Registry & Education (ASPIRE) Program is a similar registry examining the effects of the Phoenix Metropolitan Primary Stroke Center Matrix on Stroke care in Arizona. The long-term goal is to develop a plan to effectively deliver acute Stroke care to Stroke victims across Arizona.

The SHARE Program is endorsed and developed by the Arizona Department of Health Services, Bureau of Emergency Medical Services (BEMS). The SHARE initiative promotes public awareness, public education, data collection, and research of cardiac arrest. Most of all, SHARE is designed to advance research in Out of Hospital Cardiac Arrest (OHCA) and seek new strategies to save lives.

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TULAREMIA By Craig Levy

Two human cases of tularemia have been reported in Arizona in 2005. The cases involved a mother and son in Coconino County who became ill after skinning a tularemia infected rabbit. The mother developed the ulceroglandular form of tularemia (see



below), whereas the boy developed pharyngitis. Tularemia has also been identified as the cause of illness in a pet cat in Yavapai County.

Tularemia (also known as rabbit fever and deerfly fever) is a zoonotic disease caused by the gram negative coccobacillus *Francisella tularensis*. Incubation period varies from 1 to 14 days with an average of 3 to 5 days. In

Arizona, tularemia is transmitted to humans by direct contact with blood and tissues of infected rabbits and rodents (such as occurs during the skinning of game), or by deerfly bite. In other parts of the country (especially the Midwest) tularemia is transmitted by ticks. Less common modes of transmission would include animal bites, ingestion, and inhalation.

There are six clinical forms of tularemia which are determined primarily by their route of inoculation.

1) The most common clinical presentation is ulceroglandular tularemia which occurs through direct contact and insect bite exposures. Indolent ulcers develop at the site of inoculation which is commonly on the hands of hunters or any exposed skin surface in case of insect bite. Regional lymphadenopathy develops within a few days of ulceration. Other symptoms include fever, headache, chills and weakness. Bubonic plague should be included in the differential diagnosis of any patient who presents with these symptoms and has animal or insect bite exposure.

2) The glandular form of tularemia is characterized by lymphadenopathy and fever, but no skin ulcer.

3) Oropharyngeal tularemia is acquired through ingestion of bacteria in meat or water and is characterized by severe pharyngitis, abdominal pain, vomiting and diarrhea.

4) Oculoglandular infection may occur when bacteria are rubbed into the eyes, causing painful purulent conjunctivitis with regional lymphadenitis.

5) The typhoidal form may occur through inhalation of aerosolized bacteria, or possibly through gastrointestinal or intradermal inoculation. It is characterized by fever, prostration and weight loss, without lymphadenopathy. Pneumonia is often associated with typhoidal tularemia, and radiographic evidence may show evidence of mediastinal lymphadenopathy.

6) The pleuropulmonary form is characterized by pneumonia.

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Rubella--Not Gone But Often Forgotten

Karen Lewis, M.D.

Rubella is a viral disease that has almost disappeared in the United States since the rubella vaccine was licensed in 1969. In 2004, there were only nine rubella cases reported in the United States. Arizona has not had a reported case of rubella since 2000. However, a recent outbreak of rubella in eastern Canada underscores the importance of continuing to give rubella vaccine to children and adults in Arizona.

Canadian outbreak 2005

In September 2004, cases of rubella started to be seen in the Netherlands in a group of people opposed to vaccines. It is believed that travel between the Netherlands and Canada was the link that brought rubella to Canada. As of May 30, 2005, there have been 258 documented cases of rubella in southwestern Ontario, Canada. Seven of the confirmed cases have been in pregnant women, and another 15 pregnant women may have been exposed.

Clinical presentation

Rubella can be difficult to diagnose clinically. Twenty-five to fifty percent of cases are asymptomatically infected. Patients infected with rubella are nontoxic. In children there is usually not a prodrome. In adolescents and adults there may be a few

days of low grade fever and mild upper respiratory symptoms. Then a discrete maculopapular rash starts on the face and travels down the body. By the second day, the rash begins to disappear from the face. By the end of the third day the rash has usually disappeared. There may be enlargement of the posterior occipital, post auricular, and cervical lymph nodes.

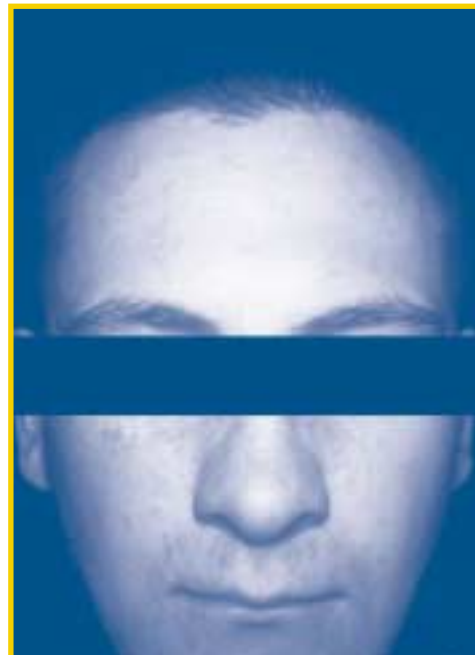
Complications

Rubella is a benign disease unless a pregnant woman becomes infected. Maternal rubella during pregnancy can result in miscarriage, fetal death, and congenital abnormalities including microcephaly, mental retardation, cataracts, glaucoma, deafness, patent ductus arteriosus, hepatosplenomegaly and purpuric skin lesions.

During 1964 and 1965 a rubella epidemic in the United States caused an estimated 12.5 million cases of rubella and 20,000 cases of congenital rubella syndrome (CRS) which led to more than 11,600 babies born deaf, 11,250 fetal deaths, 2,100 neonatal deaths, 3,580 babies born blind and 1,800 babies born mentally retarded.

Vaccination

Rubella is prevented by vaccination. Although there is a monovalent rubella vaccine available, rubella vaccination is usually given as part of a combined vaccine against measles, mumps, and rubella (MMR). The first MMR is administered to children on or after their first birthday, with the second dose usually given at 4-6 years of age. People born in 1957 or later



Patient with face rash due to rubella

should be considered susceptible to rubella unless they have had at least one dose of rubella vaccine or have serologic documentation of immunity.

Rubella vaccine is contraindicated in pregnant women. However, the Centers for Disease Control and Prevention evaluated hundreds of infants born to women who did inadvertently receive rubella vaccine during pregnancy, and found that there was no evidence of congenital abnormalities.

Serologic Diagnosis

The evaluation of a rash illness for postnatally acquired rubella is done by testing for rubella-specific IgM and IgG. A positive IgG and a negative IgM indicates immunity to rubella, arguing against rubella as the cause of the rash. A positive IgM and a negative IgG is compatible with acute rubella, although false positive IgMs can occur. Acute rubella can also be diagnosed by a four-fold rise in IgG titer between acute and convalescent sera.

Karen Lewis, M.D. is the Medical Director, Bureau of Epidemiology and Disease Control



Infant with congenital rubella syndrome

Bordetella pertussis On the Ropes

Will Humble

The vaccination tools are finally here to put an end to the increase in pertussis in the U.S. and Arizona. The Food and Drug Administration (FDA) approved two new vaccines to help fight pertussis this spring. The GlaxoSmithKline vaccine, called Boostrix™ is approved for adolescents from 10 to 18 years old. The sanofi pasteur vaccine, called ADACEL™ is approved for adolescents and adults from 11 to 64 years old.

Pertussis immunity from the childhood vaccine series wears off within a few years, and up until now, there has been no vaccine for anybody over the age of 6 to protect against pertussis. As a result, adolescents and adults have been susceptible to the disease and have been transmitting the disease to infants less than 6 months of age, who represent 80% of the deaths from pertussis.¹

Despite high rates of pertussis vaccination among children less than 7, pertussis cases in the U.S. have increased from 1,248 in 1981 to an annual average of 9,431 between 1996-2003.² In 2003, there were 11,647 cases of pertussis, the highest number reported since 1964.³

From January through June, more than 500 cases of pertussis have been reported in Arizona. Approximately 18% of the cases have been among infants less than 1 year old. Approximately 41% of the cases have been in high school and middle school students. Adults have represented approximately 25% of the pertussis cases. See Figure 1.

A study by Bisgard et.al. (2004) found that family members were the source of 75% of infant pertussis infections. Of all of the sources of infection with known age, 56% were adults over 19 years old, 20% were age 10-19, 17% were age 0-4 years old, and 7% were age 5-9 years old.⁴ See Figure 2.

These new vaccines will help us implement important public health interventions that will put pertussis on the ropes. First, we need to make sure

that all family members and caregivers of newborns are immunized with the new pertussis booster vaccine prior to delivery. Secondly, we need to begin incorporating the new vaccines into the routine vaccination series for adolescents and adults as soon as possible.

The new vaccines contain acellular components of *Bordetella pertussis*, diphtheria toxoid, and tetanus toxoid. Boostrix™ can be used in adolescents in place of tetanus and diphtheria toxoids (Td). ADACEL™ can be used as a replacement for the current Td booster with administration every 10 years until age 64. The new vaccines are not indicated in people who have been fully vaccinated against tetanus and diphtheria within the last 5 years due to an increased incidence and severity of local adverse reactions. The Arizona Department of Health Services has recently waived the school entry Td booster requirement for adolescents until October 31, 2005 in order to encourage the use of the new vaccines for school entry.

These new pertussis booster shots will become available through the Vaccines for Children program as soon as the Centers for Disease Control and Prevention (CDC) concurs with the new Advisory Committee on Immunization Practices recommendations for these vaccines and the CDC negotiates a price for the vaccines with the manufacturer.

Figure 1

Arizona Pertussis Cases by Age January - June 2005 (n=504)

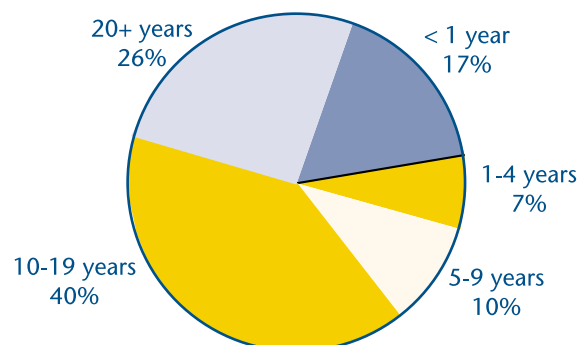
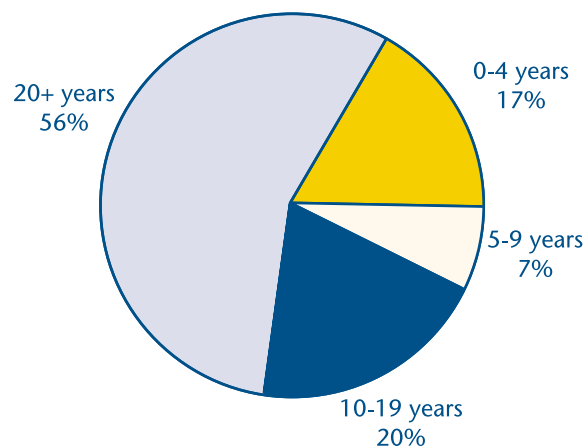


Figure 2

Source of Infant Pertussis Infection by Age Bisgard et.al. 2004 (n=264)



Once these new vaccines become incorporated into routine vaccinations for adolescents and adults we will be on our way to eliminating another infectious disease as a public health hazard during your career.

1. Vitek, Pascual, Baughman, Murphy. Increase in deaths from pertussis from young infants in the United States in the 1990s, *Pediatr Infect Dis J*. 2003;22:628-634.6.
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Noteworthy

2005-2006 Influenza Season Partnerships are key to success!

On August 5th, ADHS will invite influenza vaccine providers and other interested persons to attend a follow-up session to the Governor's Influenza Vaccine Summit held in March. The session will provide updates perspective from last year's influenza season, updates on this year's influenza vaccine supply, and discussion of strategies to vaccinate individuals at highest risk of complications from influenza (ie: children and the elderly), their household contacts, and the health care workers.

The Governor's Influenza Vaccine Summit successfully brought together vaccine providers, the Arizona Department of Health Services, and County Health Departments to form partnerships to meet the needs of the

state and each community. This session will be a continuation of the first meeting, providing the most recent information to develop strategies for the 2005-2006 influenza season.

If you would like to be invited, please contact Judy Sorce-Bauman at 602.364.3638 or email sorcej@azdhs.gov.

Sun Safety

Educators in all Arizona's k-8 public and charter schools will be teaching children the importance of sun safety, thanks to the Arizona Department of Health's SunWise program and the state's new skin cancer education law.

On August 13, educators will begin incorporating the SunWise School Program into their existing curriculum. Already, 650 of Arizona's 1,049 k-8 schools are enrolled in the

program. Passage of the bill will provide the remaining schools access to the free program.

Sun safety is extremely important for Arizonans because our state ranks No. 2 in the world in skin cancer incidence rates. (Australia is No. 1). Since 80 percent of a person's lifetime exposure to the sun occurs within the first 18 years, it's crucial to protect and educate children to reduce skin cancer rates.

Private schools can also register to receive the free SunWise program but are not required to teach it by law. For more information, contact the Arizona Department of Health Services SunWise program at www.azdhs.gov/phs/sunwise or program coordinator Sharon McKenna at mckenns@azdhs.gov. An expanded website and resources will be available on-line by August 1.

TULAREMIA continued from page 3

Tularemia cases are relatively uncommon in Arizona with seven cases being reported during the last ten years (1995-2004). To date, all of the human cases of tularemia in Arizona have been exposed in areas above 3,000 feet in elevation, and as a consequence exposure risk is considered to be greater in the northern and eastern counties. However, in year 2000 tularemia was identified as the cause of morbidity and mortality in certain animals in a zoological park in Maricopa County. As a result, health providers cannot rule-out the potential for infection in lower elevations. The strain of tularemia identified in the zoological park was type B or subsp. *holarctica* which is not the endemic strain usually found in Arizona (type A or subsp. *tularensis*).

Tularemia is a reportable disease, and prompt reporting of suspect cases is strongly advised due to similarities of this disease to plague and its potential for use as an agent of bioterrorism. Plague should be included in the differential diagnosis, and infection control measures appropriate for pneumonic plague (droplet precautions) should be implemented to prevent secondary transmission.

Laboratory diagnosis can be done by direct fluorescent antibody (DFA) test on lymph node aspirate or wound lesion, and PCR can be done on whole blood or culture. Laboratory diagnosis can also be made serologically by demonstrating a four-fold rise in specific antibody between acute and convalescent sera. *F. tularensis* can be cultured on special media (ex. cysteine-glucose agar) from lesion or node aspirate material, or less likely from blood or sputum (depending on symptoms). Diagnostic support for serology and culture is available through the Arizona State Health Laboratory.

Antimicrobial choices for the treatment of tularemia are streptomycin, gentamicin, tetracyclines and chloramphenicol.

Tularemia infection can be prevented by doing the following:

1. Avoid direct contact with sick animals, especially rabbits.
2. Wear rubber gloves when skinning game.
3. Thoroughly cook wild game meat.
4. Avoid drinking untreated water.
5. Avoid bites from deerflies and ticks through proper use of insect repellents and protective clothing in areas where vectors occur.

For more information on tularemia, contact Vector-Borne and Zoonotic Diseases Program staff at .602.364.4562.

Craig Levy is ??? and can be reached at 000.000.0000 or clevy@azdhs.gov.



SUMMARY OF SELECTED REPORTABLE DISEASES

Year to Date (January - June, 2005)^{1, 2}

| | Jan - June 2005 | Jan - June 2004 | 5 Year Median Jan - June |
|---|--------------------|--------------------|-----------------------------|
| VACCINE PREVENTABLE DISEASES: | | | |
| <i>Haemophilus influenzae</i> , serotype b invasive disease (<5 years of age) | 1 (0) | 0 (0) | 4 (1) |
| Measles | 1 | 0 | 0 |
| Mumps | 0 | 0 | 1 |
| Pertussis (<12 years of age) | 263 (129) | 70 (39) | 65 (39) |
| Rubella (Congenital Rubella Syndrome) | 0 (0) | 0 (0) | 0 (0) |
| FOODBORNE DISEASES: | | | |
| Campylobacteriosis | 457 | 385 | 324 |
| <i>E.coli</i> O157:H7 | 15 | 8 | 12 |
| Listeriosis | 4 | 5 | 6 |
| Salmonellosis | 288 | 294 | 282 |
| Shigellosis | 152 | 192 | 179 |
| VIRAL HEPATITIDES: | | | |
| Hepatitis A | 106 | 145 | 169 |
| Hepatitis B: acute | 197 | 116 | 92 |
| Hepatitis B: non-acute | 577 | 572 | 572 |
| Hepatitis C: acute | 0 | 0 | 4 |
| Hepatitis C: non-acute (confirmed to date) | 4,161 (1,663) | 5, 674 (1,900) | 4, 408 (1,922) |
| INVASIVE DISEASES: | | | |
| <i>Streptococcus pneumoniae</i> | 454 | 414 | 526 |
| <i>Streptococcus</i> Group A | 167 | 147 | 124 |
| <i>Streptococcus</i> Group B in infants <90 days of age | 34 | 24 | 17 |
| Methicillin-resistant <i>Staphylococcus aureus</i> ³ | 725 | N/A | N/A |
| Meningococcal Infection | 30 | 6 | 18 |
| SEXUALLY TRANSMITTED DISEASES: | | | |
| Chlamydia | 9,896 | 7,136 | 7,235 |
| Gonorrhea | 2,141 | 1,843 | 1,971 |
| P/S Syphilis (Congenital Syphilis) | 71 (12) | 83 (23) | 89 (13) |
| DRUG-RESISTANT BACTERIA: | | | |
| TB isolates resistant to at least INH (resistant to at least INH & Rifampin) | 9 (0) | 9 (2) | 7 (0) |
| Vancomycin resistant <i>Enterococci</i> isolates | 1,099 | 666 | 531 |
| VECTOR-BORNE & ZOONOTIC DISEASES: | | | |
| Hantavirus Pulmonary Syndrome | 3 | 1 | 1 |
| Plague | 0 | 0 | 0 |
| West Nile virus Infection | 1 | 143 | N/A |
| Animals with Rabies ⁴ | 99 | 37 | 42 |
| ALSO OF INTEREST IN ARIZONA: | | | |
| Coccidioidomycosis | 1,411 | 1,614 | 1,099 |
| Tuberculosis | 111 | 103 | 90 |
| HIV | 346 | 232 | 232 |
| AIDS | 281 | 231 | 239 |

¹ Data are provisional and reflect case reports during this period.

² These counts reflect the year reported or tested and not the date infected.

³ MRSA was not reportable before October 2004.

⁴ Based on animals submitted for rabies testing.

Data compiled by Offices of Infectious Disease and Office of HIV/AIDS Services



Prevention bulletin



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